U.S. Department of Labor

Office of Administrative Law Judges
O'Neill Federal Building - Room 411
10 Causeway Street
Boston, MA 02222

(617) 223-9355
(617) 223-4254 (FAX)

Issue Date: 03 March 2005

CASE NO.: 2003-LHC-02480

OWCP NO.: 01-156876

In the Matter of

DONALD CHAPPELL (Deceased) and
ELEANOR RUTH CHAPPELL (Widow)
Claimants

v.

ELECTRIC BOAT CORPORATION
Employer/Self-Insured

Appearances:

Stephen C. Embry (Embry & Neusner),
Groton, Connecticut for the Claimant

James T. Hornstein (Higgins, Cavanagh & Cooney),
Providence, Rhode Island for the Employer

Before: Daniel F. Sutton, Administrative Law Judge

DECISION AND ORDER AWARDING BENEFITS

I. Statement of the Case

This proceeding arises from claims for worker's compensation and death benefits filed by Donald Chappell and his widow, Eleanor Ruth Chappell (hereinafter the "Decedent" and the "Widow" or collectively, the "Claimants"), against the Decedent's former employer, the Electric Boat Corporation (EBC), under the Longshore and Harbor Workers' Compensation Act, as amended, 33 U.S.C. § 901, *et seq*. (the LHWCA). The Decedent worked for EBC from 1962 to 1986 at EBC's shipyard in Groton, Connecticut where he was exposed to asbestos, industrial dust and welding and paint fumes. After his retirement in 1986, he developed chronic obstructive lung disease and lung cancer from which he died on November 20, 2003. The Decedent and Widow claim that the Decedent's lung disease and death were caused by his employment at EBC, and they seek an award of compensation, death benefits, medical care and attorney's fees. EBC counters that the Decedent's lung disease and death were unrelated to his employment. The parties were unable to resolve the claims through informal proceedings before

the Office of Workers' Compensation Programs, and that office transferred the case to the Office of Administrative Law Judges for a formal hearing on the claims.

Pursuant to notice a hearing was conducted on January 27, 2004 in New London, Connecticut. The Widow appeared with counsel and testified, and an appearance was made by counsel on behalf of EBC. Documentary evidence was admitted as Claimant's Exhibits ("CX") 1-17 and EBC Exhibits ("EX") 1-4. The record was then closed, and the parties were allowed until March 15, 2004 to file briefs. Thereafter, the Widow moved to reopen the record to receive medical reports from two of the Decedent's treating physicians, asserting that the doctors had been asked to submit the reports prior to the hearing but did not respond in time to permit their reports to be offered at the hearing. EBC objected on the ground that the doctors had not been identified as potential witnesses prior to the hearing and because the Claimant never indicated at the time that the record was closed that any additional medical reports were anticipated. By order issued on February 27, 2004, the Widow's motion was denied based on the Court's finding that she had not shown that reopening was warranted by extraordinary or compelling reasons. *See Smith v. Ceres Terminal*, 9 BRBS 121, 124 (1978). Both parties then filed briefs.

After careful analysis of the evidence contained in the record, and after consideration of the parties' arguments, I conclude that the Claimants have established entitlement under the LHWCA to disability compensation, medical benefits, funeral expenses, survivor's benefits, interest on unpaid compensation and expenses, and attorney's fees. My findings of fact and conclusions of law are set forth below.

II. Stipulations and Issue Presented

At the hearing, the parties offered the following stipulations: (1) this matter arises under the jurisdiction of the LHWCA; (2) there was an employer-employee relationship between the Decedent and EBC at all material times; (3) all filing, notice, claim and controversion provisions of the LHWCA were timely met; (4) the applicable National Average Weekly Wage for computation of any benefits awarded is $498.27; (5) Eleanor Chappell is the widow and presumptive dependent of Donald Chappell; and (6) the Decedent had a 100% lung impairment as of September 24, 2002 when he was diagnosed with lung cancer. Hearing Transcript ("TR") at 9-10. The parties further stipulate that the only issue presented for adjudication is whether the Decedent's lung disease and death were causally related to his employment at EBC. *Id.*

III. Findings of Fact and Conclusions of Law

A. Background

The Decedent was born in 1924, and he died on September 20, 2003 at the age of 79. CX 6. He testified at a deposition taken on October 24, 2002. CX 5. He served in the Air Force during World War II after graduating from high school, and he returned to school after the war, studying refrigeration and heating at the New England Technical Institute in Providence. CX 5

at 4-5. He married Eleanor Ruth Allen in 1947, and he testified that she was his only dependent at the time of his deposition. *Id.* at 25-26; CX 7.

After completing trade school, the Decedent was employed for five years as a service man for a heating and oil contractor. CX 5 at 5. He testified that he installed and removed boilers which were covered with asbestos insulation. *Id.* at 6. He then worked until 1962 in his own plumbing and heating business . *Id.* at 6-7. He said that he was not exposed to asbestos as much in this line of work, but he estimated that he still had to work on asbestos-insulated boilers a couple of times a month. *Id.* at 7.

In 1962, the Decedent was hired by EBC to work as a pipe fitter at EBC's submarine shipyard which is adjacent to the navigable Thames River in Groton, Connecticut. *Id.* at 8. The Decedent testified that he was exposed to asbestos while working as a pipe fitter because the piping that he installed was covered with asbestos. *Id.* at 9-10. Although the only asbestos material that he personally handled was found in gaskets that he used for fittings in pipes, the Decedent said that he worked within five to ten feet of pipe laggers who installed asbestos insulation on pipes. *Id.* at 10-12. He testified that there was visible dust from asbestos, welding and grinding in the air aboard the submarines where he worked. *Id.* at 11, 13. He also said that he was exposed to visible smoke, fumes and dust from welders, burners and grinders to whom he worked in close proximity as a pipe fitter. *Id.* at 13-14.

After three or four years working as a pipe fitter, the Decedent was promoted to an Inspector's position in EBC's Nuclear Inspection Department where he was responsible for overseeing construction and testing of nuclear systems. *Id.* at 15. He testified that while working as an inspector, he was exposed to airborne asbestos as he had to inspect pipes and systems that were insulated with asbestos. *Id.* at 16, 18. He also said that he was exposed as an inspector to welding and painting fumes as well as burning and grinding dust while aboard submarines. *Id.* at 17-18. In 1968, the Decedent was promoted to the position of foreman in the inspection department. *Id.* at 15. As a foreman, the Decedent went aboard submarines once or twice daily to oversee the inspectors under his supervision, but he had much less interaction and contact with other trades and estimated that 60 percent of his time was spent in offices and meetings off of the submarines. *Id.* at 30-33. However, he testified that he was exposed to asbestos and grinding dust and paint fumes when he went aboard submarines as a foreman, and he said that he observed dust in the air when he went aboard submarines undergoing overhaul and asbestos removal up until the early 1970s when EBC began to take "more caution." *Id.* at 18-20. The Decedent further testified that at some point in the 1980s, there was a greater awareness of the possible dangers associated with asbestos exposure, and EBC started stressing the importance of employees wearing protective equipment including face masks and having work with asbestos done while other employees were not present. *Id.* at 34-35. The Decedent's account of his exposure to asbestos dust, welding fumes and grinding dust at EBC is corroborated in material respects by the testimony of a co-worker, Arthur Delaney. CX 11. There is no contrary evidence.

In addition to the occupational exposure to asbestos, welding and paint fumes and grinding dust, the Decedent testified that he smoked at least a package of cigarettes per day from approximately 1942 when he went into the military service to 1995 when he quit smoking after suffering an episode of bronchitis. CX 21-22, 40-41. Mrs. Chappell, who was married to the Decedent for 54 years, confirmed that he smoked cigarettes during their marriage but stopped in approximately 1995 after he suffered a severe attack of bronchitis while in Florida. TR 28, 31.

The Decedent retired from EBC in June of 1986 after he was injured in a non-occupational automobile accident. CX 5 at 22. Subsequent to his retirement, the Decedent experienced worsening respiratory symptoms. *Id.* at 22, 23. He sought treatment from his primary care doctor and was eventually referred to Leon Puppi, M.D. *Id.* at 37; CX 2.

B. Medical Records

Records from Dr. Puppi reflect that he began treating the Decedent in September of 1998. CX 2. In a report dated September 21, 1998, Dr. Puppi reported that spirometic testing showed a severe obstructive ventilatory impairment and that a CT scan of the chest revealed large bullae in the apices with calcified pleural plaques, "suggesting asbestos exposure." *Id.* at 36. Dr. Puppi stated that the Claimant's clinical history is consistent with "moderate-to severe chronic obstructive pulmonary disease secondary to a profound history of cigarette smoking." *Id.* He also stated that the CT scan revealed "significant bullous emphysema with calcified pleural plaques, consistent with asbestos exposure." *Id.*

The Decedent underwent a series of tests in May of 2000. A chest x-ray on May 8, 2000 was interpreted as showing fibrous emphysema and "a soft tissue density in the right apex [which] may be severe pleural thickening." *Id.* at 28. A CT scan on May 17, 2000 was interpreted as showing "complex findings" including "significant apical pleural thickening and increased soft tissue scarring and bullous change in the right apex" and "diffuse areas of fibrosis and peripheral pleural thickening and nodularity particularly at the left base." *Id.* at 27. A pulmonary function test on July 31, 2000 revealed a normal total lung capacity, increased residual volume indicating air trapping and decreased forced expiration consistent with an obstructive ventilatory defect. *Id.* at 25. The interpreting physician, James McCormack, M.D. also reported that there was no significant improvement in the indices of forced expiration after bronchodilator treatment and that the Decedent's diffusing capacity was decreased. *Id.* Dr. McCormack's impression was a severe obstructive ventilatory defect and a severe gas transport defect. *Id.* Dr. McCormack also had the decedent undergo a pulmonary exercise stress test on August 1, 2000 which he interpreted as consistent with severe chronic obstructive pulmonary disease, cardiopulmonary deconditioning and exercise-induced hypoxemia.[1] *Id.* at 20-21. Follow-up CT scans in July and August of 2002 again were positive for changes consistent with chronic underlying lung disease with pleural plaques, linear fibrotic changes and pleural thickening suggestive of asbestos exposure and a nodular opacity at the left lung base that was

[1] Hypoxemia is a reduction of oxygen supply to tissue below physiological levels despite adequate perfusion of the tissue by blood. Dorland's Illustrated Medical Dictionary (28th Ed. 1994) at 812.

suspicious for malignancy. *Id.* at 12-16. Unfortunately, this suspicion was confirmed on November 7, 2002 when a needle biopsy was taken of the left lower lobe abnormality, producing a pathology diagnosis of a non-small cell carcinoma with features consistent with a poorly differentiated adenocarcinoma. CX 9 at 23-25.

The Decedent underwent radiation treatment but was later found to have metastases to the brain leading to a period of hospitalization in September of 2003 for neurological weakness. CX 9 at 2-9. At that time, Dr. Puppi reported that the Decedent's diagnoses were (1) a cerebrovascular accident by history most likely due to a right parietal mass, (2) Stage IV adenocarcinoma of the lung with brain metastasis, status post lung and brain irradiation, (3) advanced oxygen-dependent bullous emphysema secondary to a profound history of cigarette smoking with very limited exercise tolerance, and (4) malnutrition by history. *Id.* at 7. He was discharged from the hospital and expired at home on September 20, 2003. John J. Bossian, D.O., who was the Decedent's attending physician during his final hospitalization, certified the Decedent's death certificate on which he listed respiratory failure as the immediate cause of death with COPD of seven to ten years' duration and lung cancer with brain metastasis of one year's duration as underlying conditions leading to the immediate cause of death. CX 6.

C. Causation

Pursuant to section 20(a) of the LHWCA, it is presumed that a claim comes within the LHWCA's provisions. 33 U.S.C. § 920(a). This statutory presumption applies to the connection between and employee's injury or disease and his or her employment. *Swinton v. J. Frank Kelly, Inc.*, 554 F.2d 1075, 1082 (D.C. Cir. 1976), *cert. denied*, 429 U.S. 820 (1976). To invoke the presumption, there must be a *prima facie* claim for compensation which "must at least allege an injury that arose in the course of employment as well as out of employment." *U.S. Industries/Federal Sheet Metal, Inc., et al., v. Director, OWCP*, 455 U.S. 608, 615 (1982). A claimant presents a *prima facie* case by establishing (1) that he or she sustained physical harm or pain and (2) that an accident occurred in the course of employment or conditions existed at work, which could have caused the harm or pain. *Clophus v. Amoco Prod. Co.*, 21 BRBS 261, 265 (1988).

The Claimants have alleged that the Decedent's lung disease and death were, at least in part, caused by his employment, and they rely on medical opinions from Drs. Pohl and Daum, which are discussed below, to establish the required nexus between the Decedent's fatal lung condition and his employment at EBC. EBC concedes that the Claimants have established a *prima facie* case. EBC Brief at 32. Consequently, EBC is required to produce "substantial evidence" severing the presumed connection between the Decedent's lung disease and death and his working conditions at EBC. *Volpe v. Northeast Marine Terminals*, 671 F.2d 697, 701 (2nd Cir. 1981). EBC's evidence must not only show that the Decedent's lung disease was not *caused* by his employment; it must also demonstrate that the lung disease was not *aggravated* by employment. *See Bath Iron Works Corp. v. Director, OWCP*, 109 F.3d 53, 56 (1st Cir. 1997).

EBC contends that it has introduced sufficient evidence, consisting of medical opinions from Drs. Pulde, Kern and Teiger, to rebut the presumed causal connection and demonstrate that the Decedent's lung disease and death were not caused, aggravated or accelerated by his occupational exposures at EBC. EBC Brief at 32. The Claimants question whether EBC's evidence is sufficient to overcome their *prima facie* showing of causation, and they argue that the weight of the evidence supports a finding that the Decedent's lung disease and death were causally related to his employment at EBC. Claimants' Brief at 15-16. In my view, the evidence introduced by EBC is sufficient to rebut the presumption. Drs. Pulde, Kern and Teiger all discussed the Decedent's work history and concluded that his lung cancer was caused by cigarette smoking, that his death was caused by the lung cancer and that occupational exposures did not cause or contribute to his lung cancer or death. If these opinions are credited, causation would not be established. Thus, EBC's evidence qualifies as "substantial" because it is the kind of evidence that a reasonable mind might accept as adequate to support a conclusion. *Richardson v. Perales*, 402 U.S. 389, 401 (1971). While the Claimants do raise some valid questions about the opinions from Drs. Pulde, Kern and Teiger, I find that these opinions are not so hedged or speculative to deprive them of any probative weight. *Cf. American Grain Trimmers, Inc. v. Office of Workers' Compensation Programs,* 181 F.3d 810, 818 (7th Cir. 1999) (affirming ALJ finding that employer did not introduce substantial evidence where sole evidence opposing causal relationship was a doctor's statement that he was confident that worker's fatal heart attack was not related to his job even though the doctor conceded he had no idea what the worker was doing before suffering the heart attack). *See also Ortco Contractors, Inc. v. Charpentier*, 332 F.3d 283, 289 (5th Cir. 2003) (rejecting requirement that an employer "rule out" causation or submit "unequivocal" or "specific and comprehensive" evidence to rebut the presumption and reaffirming that "the evidentiary standard for rebutting the § 20(a) presumption is the minimal requirement that an employer submit only 'substantial evidence to contrary.'"), *cert. denied*, 540 U.S. 1056 (2003) (underlining and internal quotation marks in original). In view of the determination that EBC had introduced substantial evidence to rebut the Claimants' *prima facie* case, the presumption "falls out" of the case, and the record must be considered as a whole to determine whether the Claimants have carried their burden of establishing causation by a preponderance of the evidence. *See John W. McGrath Corp. v. Hughes*, 264 F.2d 314, 317 (2d Cir. 1959), *cert. denied*, 360 U.S. 931 (1959). Accordingly, I will weigh all of the medical opinions in the record addressing causation.

Dr. Pohl

Douglas A. Pohl, M.D., Ph.D., reviewed the Decedent's medical and occupational histories and conducted a pathology review in a report dated November 14, 2003. CX 10. Based on his review of the pathology slides, he stated that he was in complete agreement with the diagnosis of an adenocarcinoma of the left lower lobe. *Id.* at 2. He stated that 85-90 percent of lung cancers arise as a consequence of cigarette smoking and, after noting the Decedent's smoking history, stated that his past smoking likely played a role in the development of his lung cancer. *Id.* at 3. Dr. Pohl further stated that asbestos is a known carcinogen which may act as a co-carcinogen which amplifies the lung cancer risk produced by other carcinogens such as

cigarette smoke. *Id.* Dr. Pohl continued that the Decedent's asbestos exposure was significant enough to cause interstitial fibrosis and bilateral pleural plaques which were seen on his chest x-rays and which are only seen in persons who were heavily exposed to asbestos, and he added that asbestos-related lung cancer is a dose-dependent disease with higher risks occurring in individuals more heavily exposed to asbestos. *Id.* Based on the "well documented cause and effect relationship" between asbestos exposure and lung cancer, the Decedent's heavy exposure to asbestos, and the "synergistic effect" of asbestos on smoking related lung cancer risk, Dr. Pohl concluded that "it is my opinion, within a reasonable degree of medical certainty, that Mr. Chappell's occupational exposure to asbestos was a significant contributing factor to the development of his lung cancer and subsequent death." *Id.* at 4.

At his deposition, Dr. Pohl testified that he has a Ph.D. and board-certification in pathology in addition to his medical degree, and he is the Director of Pathology at the Central Maine Medical Center in Lewiston. CX 12 at 3-4, 16. He said that his practice as a pathologist has provided him with an opportunity to observe the diseases resulting from asbestos exposure such as asbestosis which is a scarring or fibrotic disease if the lung, lung cancer which arises in the bronchi and mesothelioma which is a tumor located in the lung coverings or pleura. *Id.* at 5. Dr. Pohl also said that he has had the opportunity to examine lungs of welders which contain metal materials that are present in welding fumes. *Id.* at 5-6. Regarding his consultative report on the Decedent, Dr. Pohl testified that he reviewed the Decedent's medical records and the pathology slides from the needle biopsy, and he confirmed his agreement with the original pathology diagnosis of an adenocarcinoma. *Id.* at 6-8, 21-22. He stated that the needle biopsy did not obtain a sufficiently large sample of tissue to make a determination as to whether the Decedent had asbestosis. *Id.* at 12, 22. However, he testified that asbestos exposure causes a scarring in the lungs that is distinguishable from scarring caused by emphysema, and he said that the Decedent's chest x-rays showed calcified and non-calcified pleural plaques, the corollary of which is parenchymal scarring, which is "perfectly diagnostic of asbestosis on radiological grounds. *Id.* at 13-14. Dr. Pohl was then asked to assume certain facts relating to the Decedent's occupational and smoking histories, and he responded that it is his opinion that the Claimant's exposure in EBC's shipyard was "one of the direct causes" of his lung cancer. *Id.* at 9.[2] Dr. Pohl also said that cigarette smoking contributed to the Decedent's lung cancer, and he explained that while asbestos and cigarette smoke can both independently cause lung cancer, a typical smoker such as the Decedent has a 15-fold higher risk of developing lung cancer due to long-term asbestos exposure. *Id.* at 9-10. Dr. Pohl added that had the Decedent not been exposed to asbestos, his lung cancer risk would have been substantially lower and that it is his opinion that he would not have developed lung cancer at all. *Id.* at 11. He said that asbestos exposure was a contributing factor in the Decedent's death, and he explained that a person does not have to

[2] EBC's attorney objected to the question which elicited this opinion. While I agree that Dr. Pohl was asked to assume facts regarding the Decedent's cigarette smoking (*i.e.*, that the Decedent smoked about one pack per day for 50 years until 1983 when he stopped) that are at variance with the evidence of record which indicates that the Decedent did not quit smoking until 1995, I overrule the objection since Dr. Pohl was asked to assume that the history set forth in the question "is relatively correct" and because 50 years is relatively close to the smoking history of approximately 53 years acknowledged by the Decedent. CX 12 at 9.

suffer from asbestosis before developing asbestos-related lung cancer because the level of asbestos exposure required to cause lung cancer is much lower than the level required to cause asbestosis. *Id.* at 11, 13. In this regard, he explained that asbestos exposure is similar to cigarette smoke in that half of cigarette smokers with lung cancer have no evidence of chronic obstructive pulmonary disease or emphysema, yet their lung cancer is still attributed to cigarette smoking. *Id.* at 13.

On cross-examination by EBC's attorney, Dr. Pohl stated that he did not personally review the Decedent's x-ray films and that it is his normal practice to rely on the radiology report because he is not a radiologist. *Id.* at 15-16. He agreed that scarring in the lungs may be caused by a variety of conditions, but he reiterated his opinion that the parenchymal scarring in the bases of the Decedent's lungs is consistent with asbestosis and distinguishable from the type of scarring associated with cigarette smoking, silicosis and other conditions. *Id.* at 17. Dr. Pohl also agreed that the Decedent had evidence of advanced chronic obstructive pulmonary disease and past tuberculosis, but he did not agree that the Decedent necessarily would have developed lung cancer based on cigarette smoking alone because only five percent of lifelong smokers develop lung cancer, although 90 percent of lung cancer found in American males is attributed to smoking. *Id.* at 17-19. Dr. Pohl testified that he was familiar with the theory of synergy and agreed with the proposition that smokers who are additionally exposed to asbestos have a much higher incidence of lung cancer. *Id.* at 20. Dr. Pohl said that the synergistic theory of a causal relationship between smoking, asbestos exposure and lung cancer is based on studies of smokers who were also exposed to asbestos, and he disagreed with counsel's characterization of those studies as showing that all of the individuals had asbestosis and evidence of fibrotic changes. *Id.* at 21. However, he did agree that it has been asserted by some that every individual diagnosed with cancer in the Selikoff study[3] had asbestosis which he attributed to the fact that the study covered insulators who had worked under very dusty conditions. *Id.* at 27. He also agreed that there are some types of cancer such as mesothelioma which are specifically associated with asbestos exposure and that the Decedent did not have mesothelioma. *Id.* at 20-21. Dr. Pohl testified that the synergistic relationship between cigarette smoking and asbestos in increasing the risk of developing lung cancer has been proved beyond a reasonable degree of medical certainty, that the Irvine and Bianchi studies in Scotland and Italy have shown that there are a significant number of asbestos exposed individuals who develop lung cancer without evidence of asbestosis, and that a majority of mesothelioma patients develop that form of cancer without asbestosis. *Id.* at 23-26. Finally, Dr. Pohl testified that his opinion on causation is based in part on the CT scan report of pleural and parenchymal scarring and calcified and non-calcified pleural

[3] Dr. Selikoff and colleagues published a series of lung disease in asbestos insulating workers including Selikoff, Churg, Hammond, The occurrence of asbestosis among insulation workers in the United States. Ann NY Acad Sci 1965; 132:139-155; Selikoff, Churg, Hammond, Asbestos exposure and neoplasia, JAMA 1964; 188:22-26; Selikoff, Hammond, Seidman, Mortality experience of insulation workers in the United States and Canada, 1943-1976. Ann NY Acad Sci 1979; 330:91-116; Hammond, Selikoff, Seidman, Asbestos exposure, cigarette smoking and death rates. Ann NY Acad Sci 1979; 330:473-491.

plaques which, he said, are almost always a consequence of asbestos exposure, but he conceded that one can have pleural plaques without evidence of asbestosis or lung cancer because pleural plaques are a marker of past heavy asbestos exposure which do not cause lung cancer in and of themselves. *Id.* at 27.

Dr. Daum

Susan M. Daum, M.D., a specialist in occupational medicine, conducted a review of the Decedent's medical records and reported her findings and opinions in a letter to the Claimants' attorney dated May 15, 2003. CX 1. She concluded that the Decedent developed adenocarcinoma of the peripheral lung and stated that it is her opinion that his occupational exposure to asbestos was a significant contributing factor in the development of the lung cancer and that it was probable that he had underlying pulmonary asbestosis based upon evidence of pulmonary fibrosis and a diffusion capacity deficit greater than would be expected from emphysema alone. *Id.* at 5. Dr. Daum stated that asbestos is a well-known carcinogen and that it is well-established that the incidence of all types of lung cancer is increased in persons exposed to asbestos. *Id.* She explained that asbestos-related lung cancers do not appear different, clinically or pathologically, from lung cancers appearing in the general population or those caused by other carcinogens. *Id.* Dr. Daum further stated that asbestosis is not a necessary precursor to asbestos-related lung cancer because there are different biological mechanisms by which asbestos causes pulmonary fibrosis and lung cancer. *Id.* at 6. Regarding these different mechanisms, she stated that fibrosis caused by asbestos is "mediated by a reaction of asbestos with inflammation cells causing the expression of cellular hormones (called cytokines)" while "[c]ancers are caused by asbestos fibers acting on the genetic material in the nucleus of the cell" thereby producing changes in the genetic material of the type that is known to be associated with "the initiation and promotion of the development and increasing abnormality of cancer cells." *Id.* Dr. Daum wrote that the "separate pathogenesis of pulmonary asbestosis and asbestos-associated lung cancer is confirmed by several epidemiologic studies which demonstrate that excess lung cancer occurs in asbestos-exposed populations who do not have fibrosis as measured by x-rays." *Id.* (citation of studies omitted). She further stated that lung cancer incidence in asbestos workers who smoke is five to seven times higher than the incidence among the general population of smokers who are not exposed to asbestos and 50 to 70 times higher than the rate seen in the general population of non-smokers who are not exposed to asbestos. *Id.*

Dr. Daum testified at a deposition taken on December 19, 2003. CX 13. She is board-certified in both occupational and preventative medicine, and she is a certified "B-reader" for chest x-rays.[4] *Id.* at 7-8. She has practiced occupational medicine since 1979, and she

[4] A certified "B-reader" is a physician who has demonstrated designated levels of proficiency in assessing and classifying x-ray evidence of pneumoconiosis and other diseases by successful completion of an examination administered by the Centers for Disease Control and Prevention ("CDC"), National Institute for Occupational Safety and Health ("NIOSH"). *See* 42 C.F.R. § 37.51 (2004); *Mullins Coal Co. v. Director, OWCP*, 484 U.S. 135, 146 n.16 (1987), *rehearing denied*, 484 U.S. 1047 (1988); *In Re Joint Eastern and Southern Districts Asbestos Litigation*, 237 F.Supp.2d 297, 307-308 (E.D.N.Y. 2002).

participated in a study of EBC workers that showed a slightly reduced cancer mortality rate which she attributed to workers dying earlier from pulmonary asbestosis due to concentrated asbestos exposure, rather than EBC being a less toxic work environment. *Id.* at 7, 10-11, 32. She is a member of the Department of Community and Preventative Medicine at the Mount Sinai School of Medicine, and she has published extensively on occupational lung disease. *Id.* at 7, Dep. Exhibit 1.

In her testimony, Dr. Daum explained that emphysema caused by cigarette smoking and pleural scarring, fibrosis and asbestosis caused by asbestos exposure affect the lungs differently with the former causing a loss of elastic tissue and resulting in an enlargement of lung capacity, the latter resulting in a stiffening of the lungs and a reduction in lung capacity. *Id.* at 19-21. She further explained that when a person suffers from both emphysema and fibrosis, the effects on lung capacity tend to cancel each other with the result that the person will have a normal total lung capacity but a very low diffusion capacity which is a measurement of the surface area of the lung available for gas exchange. *Id.* at 18, 22. Dr. Daum confirmed Dr. Pohl's testimony that studies have shown the synergistic relationship or "multiplicative interaction" between asbestos exposure and smoking in the development of lung cancers, and she said that a study has shown that individuals with asbestos exposure have increased cell mutations called oncogens even without fibrosis and asbestos-related lung cancer. *Id.* at 22-24. She stated that asbestos exposure can cause or promote development of all types of lung cancer and that it is her reading of the literature that although scarring may increase the risk of lung cancer, it is not necessary for cancer to develop. *Id.* at 24-26. Thus, she stated that "[y]ou don't need fibrosis to have any form of respiratory or gastrointestinal cancer." *Id.* at 29. And, she said that lung cancer requires less exposure to asbestos than asbestosis. *Id.* at 32-33. Nevertheless, she testified that she had done a "B" reading of the Decedent's May 8, 2000 chest x-ray which she interpreted as positive for abnormalities "related to the presence of both pulmonary and asbestosis and emphysema and cancer" and that her interpretation of the pre-radiation CT scan findings of fibrosis, pleural thickening and pleural scarring was "likely asbestosis." *Id.* at 38, 52-53. Dr. Daum discussed the Decedent's chronic obstructive lung disease ("COPD") and testified that inhalation of dust and many irritants including cigarette smoke, grinding dust and welding and fumes cause or contribute to COPD and emphysema. *Id.* at 34-35. In response to a question which asked her to assume the Decedent's history of exposure to asbestos, cigarette smoke, grinding dust and welding and paint fumes, Dr. Daum testified that it is her opinion that the Decedent suffered from asbestosis in the form of pulmonary fibrosis, COPD and lung cancer. *Id.* at 41.[5] She stated that she based her diagnosis of asbestosis on the presence of pleural thickening and fibrosis on the Decedent's chest x-ray, his extremely low diffusion capacity and history of exposure. *Id.* at 42, 63. She then explained that the pulmonary function testing showed that the Decedent's reduced diffusion capacity was not accompanied by an increased total lung capacity, as one would expect with COPD or emphysema alone, which indicates the presence of restrictive lung disease in addition to the COPD which is an obstructive disease. *Id.* at 45-46. Dr. Daum further

[5] There was no objection to this question.

testified that it is her opinion that the Decedent's exposure to asbestos, welding fumes, grinding dust and other lung irritants in the shipyard contributed to or accelerated his COPD, and that his cigarette smoking was also a significant contributing factor. *Id.* at 47, 49-50. She said that it is also her opinion that the Decedent's exposure to asbestos and other pulmonary factors such as welding and burning fumes in the shipyard contributed, along with cigarette smoking, to the development of lung cancer and that his employment was a significant contributing factor to his death which was caused by respiratory failure due to the combination of lung cancer and COPD. *Id.* at 54-55.

On cross examination, Dr. Daum acknowledged that most of her patients are referred to her by unions and attorneys, and she said that much of her occupational medicine practice relates to treatment of patients who believe that they have suffered an injury or death due to asbestos exposure. *Id.* at 57-58. She was questioned about Dr. Puppi's diagnosis which related the Decedent's lung disease to a profound smoking history, and she responded that despite Dr. Puppi's specialty as a pulmonologist, she is not surprised that a doctor might not be aware of the synergistic relationship between smoking and occupational exposure, and she added that Dr. Puppi may not have taken an occupational history or even considered the decedent's occupation. *Id.* at 59-60.[6] Dr. Daum also stated that Dr. Puppi did not know about the Decedent's exposure to welding fumes, commenting that she had never seen a cigarette smoking welder who did not have COPD, while only 28 percent of smokers generally develop COPD. *Id.* at 75. She agreed that smoking alone could have caused the decedent's COPD and emphysema but said that neither condition would have been as severe without the contribution of his occupational exposures. *Id.* at 66-67. She reiterated her opinion that a person does not need to have asbestosis in order to support a diagnosis of asbestos-related lung cancer, and she stated that a study of 10,000 people who had peripheral exposure to asbestos without developing asbestosis had a lung cancer rate that was three times the expected rate. *Id.* at 70-71. She further explained that asbestosis does not cause lung cancer and that asbestosis and lung cancer are separate and distinct diseases which are caused by different molecular processes. *Id.* at 81. She also stated that it is her opinion that although the Decedent could have developed lung cancer based on cigarette smoking alone, his occupational exposures to asbestos, grinding dust and other harmful lung irritants accelerated his development of lung cancer. *Id.* at 72. Lastly, she testified that the Decedent had pleural markers which do not themselves cause cancer but which are associated with an increase in lung cancer risk that is three times the expected rate. *Id.* at 77.

Dr. Teiger

Michael B. Teiger, M.D. reviewed the medical records, and he interviewed and examined the Decedent and on February 27, 2003. EX 1, Dep. Exhibit 1 (Mar. 2, 2003 Report). He concluded that the Decedent had an extensive asbestos exposure history, but he saw no convincing evidence for asbestos-related disease, noting that the most recent CT scan in January 2003 did not show diffuse fibrotic changes in both lungs to suggest asbestosis, that there were no

[6] It is noted that Dr. Puppi's reports do not contain an occupational history or discuss the Decedent's work at EBC, although he did comment that the medical evidence was suggestive of prior asbestos exposure. CX 2.

pleural plaques and no rales indicative of fibrosis and, finally, that the pulmonary function tests are entirely consistent with advanced obstructive airway disease and emphysema without evidence of restrictive changes that could be associated with a co-existing occupational lung disease. *Id.* at 5. He additionally concluded that the Decedent's lung cancer and COPD were related to cigarette smoking alone, and he stated that in the absence of "any clear asbestos-related disease, I believe it would be difficult to ascribe any additional causation of either his lung disease or his cancer to that exposure." *Id.* at 6.

Dr. Teiger testified at his deposition that he is board-certified in internal medicine and pulmonary disease, and he had treated many patients with asbestos exposure over the past 20 years. EX 1 at 4, 7. In addition, his *curriculum vitae*, which is appended to the deposition transcript, reflects that he is an assistant clinical professor of medicine in the Pulmonary Division at the University of Connecticut Health Center, and he was certified as a B-reader from 1985 to 1986. Dr. Teiger concluded that the Decedent suffered from advanced COPD, a major component of which was smoking-related bullous emphysema, and non-small cell lung cancer. *Id.* at 8, 9. He discussed the Decedent's pulmonary function test results and said that it is his opinion that they were entirely consistent with advanced obstructive lung disease and showed no evidence of any restrictive disease because the Decedent did not have reduced total lung capacity or reduction in his FEV_1 and FVC values. *Id.* at 10-11. Dr. Teiger further stated that his physical examination was negative for the presence of rales which indicates that asbestosis was not present. *Id.* at 11-12. He testified that the history that he obtained from the Decedent indicated that he was exposed to significant amounts of airborne free asbestos at EBC as well as other dusts and fumes without any specifics as to exposure to welding fumes or toxic chemicals. *Id.* at 13. He said that he found no evidence that the Decedent suffered from any other respiratory condition associated with his employment, explaining that individuals exposed to welding or other toxic fumes usually develop symptoms within a few hours or days of exposure or irritants and that there was no history of contemporaneous symptoms in either the medical records or the information provided by the Decedent. *Id.* at 13-14, 48-49. Dr. Teiger said that although the Decedent gave him no such history, he could imagine a situation where a he could have been exposed to respiratory irritants at work:

> I can envision a scenario in the workplace where he is a steam fitter and is working right next to a welder and the fumes are within an enclosed work environment such that he's breathing in the fumes and it becomes a clear respiratory irritant for him. That, of course, would be the environment he should avoid and, by all means, protect his lungs. I fail to see in my report and, therefore, he did not report to me this was the case, so it's my understanding from this particular case that those types of additional exposures did not exist to cause him respiratory difficulty.

Id. at 59. Based on the absence of evidence of asbestos-related pulmonary fibrosis or asbestosis, Dr. Teiger stated that it would be impossible for him to conclude that asbestos exposure caused, accelerated, aggravated or otherwise contributed to the Decedent's lung cancer and that it is his

opinion with a high degree of medical probability that cigarette smoking alone caused the Decedent's lung cancer, lung disease and emphysema. *Id.* at 15-17. Dr. Teiger stated that "it is a clear accepted fact . . . that asbestos and cigarette smoking have a synergistic effect in the development and acceleration of lung cancer." *Id.* at 17. However, he added that "[t]here is an opinion that exists in the medical literature that asbestos cannot be ascribed to accelerate lung cancer if there is no evidence for asbestos-related disease . . . which is to say, in the absence of asbestosis . . . asbestos cannot be considered causative or accelerating, and I tend to share that opinion." *Id.* at 17-18, 51. He similarly had no question that the Decedent's exposure to asbestos played no role in the development of his COPD or emphysema, but he said that he would have to "hedge" on whether occupational exposure to dust and welding fumes contributed to the Decedent's COPD "because I don't have any information to say that the gentleman was exposed to a significant amount of welding fumes or other toxic chemicals that may have contributed to the development of obstructive lung disease." *Id.* at 18-19, 47-48. Despite these reservations, Dr. Teiger said that based on the history of only occasional, indirect exposure to welding fumes and grinding dust that he received from the Decedent, it is his opinion that the Decedent's history "is not consistent with chronic toxic exposure that would cause emphysema or chronic obstructive pulmonary disease." *Id.* at 20.

On cross-examination, Dr. Teiger discussed the process by which body defensively responds to inhaled lung irritants through mucous and macrophage production, and he stated that it is generally believed that asbestos fibers are "entirely inert" and not responsible for development of mucous production or anything else related to the airways, while other materials found in a shipyard, such as welding fumes and grinding materials, result in increased mucous production. *Id.* at 31-32. However, he agreed that inhalation of asbestos fibers results in the lung's defensive production of macrophages which have difficulty dissolving the fibers, resulting in the development of an inflammatory process that over time causes development of fibrosis or scar tissue. *Id.* at 32-33. He concurred that asbestos fibers are absorbent and can act to transport the contaminants found in cigarette smoke deeper into the lungs. *Id.* at 33. Dr. Teiger also agreed that asbestos can be taken by the body's lymphatic systems from the lungs into the pleura or linings of the lungs which results in asbestos-related pleural plaque and thickening, and he said that it is possible for a person to develop mixed lung disease with emphysema, COPD, bronchitis, asbestos-related scarring of the lungs and pleura, and lung cancer, which makes it more difficult to determine which particular disease process is affecting air flow as measured in a pulmonary function test. *Id.* at 36-38. He further agreed with a study that showed that 25 percent of asbestos-exposed individuals with normal chest x-rays had evidence of fibrosis on pathology examination. *Id.* at 40. He explained that a chest x-ray can miss fibrosis and that high resolution CT scans are superior to chest x-rays but are still not as good as a microscope. *Id.* Under redirect examination, Dr. Teiger testified that the Decedent's chest x-rays and CT Scans showed classic findings of emphysema with multiple bullous formations in both lungs, and he stated that that the pulmonary fibrosis seen on the films could be explained by the Decedent's radiation treatment and was "fairly atypical" for asbestosis or asbestos-related pulmonary fibrosis which is typically a diffuse and bilateral process. *Id.* at 52-53.

Dr. Pulde

Milo Pulde, M.D. reviewed the Decedent's medical records and issued a report on his findings and opinions on March 22, 2003. EX 4. He also reviewed the report from Dr. Teiger. *Id.* at 5-6. Based on his review of the medical evidence and literature relating to tobacco use and tobacco-related lung disease, Dr. Pulde stated that it is his opinion to a reasonable degree of medical certainty that the Decedent's COPD and lung cancer were "direct and exclusive" consequences of long-term tobacco use and that there is no evidence that his employment at EBC or any workplace exposure to asbestos contributed to either condition. *Id.* at 7. He stated that there is no evidence that the Decedent had developed parenchymal asbestos or fibrosis of the lungs secondary to asbestos exposure, and he stated that a diagnosis of asbestosis is based on: (1) a reliable clinical history of exposure to friable asbestos dust with the appropriate latency period between exposure and detection; (2) radiographic findings consistent with asbestosis as defined by the I.L.O. Classification of Radiographs; (3) pulmonary function tests demonstrating interstitial lung disease with a restrictive pattern of reduced diffusion capacity and decreased total lung capacity; (4) high-resolution CT scan findings consistent with interstitial lung disease; (5) physical examination revealing bilateral crackles at the posterior lung bases; and (6) exclusion of non-asbestos related causes of interstitial lung disease. *Id.* at 9-10. Dr. Pulde wrote that since the evidence establishes that the Decedent's lung cancer was the "direct and exclusive" result of tobacco use, "it is not necessary to implicate an additional co-carcinogen, either occupational or non-occupational, to explain its development." *Id.* at 12. In addition, he stated that since the Decedent did not fulfill the objective criteria for a diagnosis of asbestosis, "he failed to fulfill the prerequisite for the attribution of his tobacco related lung cancer in part to asbestos exposure." *Id.* He based this opinion a 1996 study which concluded that the synergism between asbestos exposure and lung cancer in actuality is a synergism between asbestosis and lung cancer. *Id.* at 13, citing Jones RN, Asbestos Exposure, Asbestos Attributed Lung Cancer, Thorax 1996; 51:9-4. He further stated that it is generally agreed that lung fibrosis or parenchymal asbestosis is a perquisite for attributing lung cancer in whole or in part to asbestos exposure, and he asserted that the Decedent's pleural plaques were more likely related to post-inflammatory pleural thickening rather than asbestos exposure. *Id.* at 13-14. Moreover, he stated that even if it is assumed that the Decedent's pleural plaques are related to asbestos exposure, the presence of such plaques, in the absence of asbestosis, would not have contributed to the Decedent's COPD or increased his risk of developing lung cancer. *Id.* at 16.

Dr. Pulde's deposition was taken on January 13, 2004. EX 3. He testified that he had been board-certified in internal medicine since 1981 and is engaged in the full-time practice of internal medicine at the Brigham and Women's Hospital in Boston. *Id.* at 4-5. He said that based upon his review of the Decedent's medical records and the literature relating to asbestosis and asbestos-related lung cancers, it is his opinion that there is no evidence that asbestos exposure caused, contributed to, accelerated or influenced the natural history or outcome of the Decedent's lung cancer, or that asbestos exposure accelerated his death. *Id.* at 9-10. Dr. Pulde testified that before he would attribute the Decedent's lung cancer, at least in part, to his asbestos exposure, he would have to see pulmonary function tests that demonstrated a restrictive lung

disorder with a decrease in diffusion capacity, chest x-rays that demonstrate findings consistent with the I.L.O. criteria for asbestosis, and chest CT findings consistent with paranchymal asbestosis, which he did not find in his review of the medical evidence. *Id.* at 11-12, 14. Dr. Pulde found that the claimant had evidence of probable post-inflammatory pleural thickening and a right pleural plaque, which he attributed to the Decedent's past history of spontaneous pneumothorax, pneumonia, tuberculosis and intra-thoracic surgery, but no definitive evidence of asbestos-related pleural plaques. *Id.* at 14, 16-17. He further stated pleural plaques are the most common manifestation of asbestos exposure, but he said that pleural plaques are simply a marker for asbestos exposure which are not synonymous with asbestosis and which do not increase the risk of developing malignancies. *Id.* at 14-15.

On cross-examination, Dr. Pulde acknowledged that he had not personally reviewed the Decedent's chest x-rays and instead relied upon the radiologists' reports, and he said that he did not have enough information to form an opinion on the cause of death. *Id.* at 19-21. He agreed the Decedent met the asbestos exposure criterion for a diagnosis of asbestosis but said that it was difficult for him to determine from the Decedent's history "[w]hether he exceeded the threshold of 25 fiber years" for asbestos-related lung cancer. *Id.* at 23-25. He also agreed that if the Decedent had a history of sufficient exposure to asbestos, a chest x-ray classified as 1/1 under the I.L.O. criteria, asbestos-related pleural plaques, and pulmonary function tests showing restrictive disease and reduced diffusion capacity, there would be "a possibility" that he has asbestosis. *Id.* at 29. However, he disagreed that the Decedent's lung cancer and the radiation treatment would mask evidence of asbestos-related scarring on the x-rays and CT scans because asbestos-related changes are diffuse while lung cancer tends to be a focal process. *Id.* at 34-35. Dr. Pulde described how inhaled asbestos is understood to result in cellular changes that can ultimately lead to the development of lung cancer in a susceptible individual, and repeated his opinion that the "presence of fibrosis remains the prerequisite for the ultimate development of the cancer." *Id.* at 45. Dr. Pulde was also questioned about the Decedent's COPD and said that he assumed that he was exposed to a variety of particulates, gasses, fumes, vapors, and ozone, certain oxides and other by-products of the welding process, but he said that such exposures do not result in any long-term airway obstruction and certainly did not result in the Decedent's emphysema. *Id.* at 58-63. Lastly, he agreed that it is possible to look at pulmonary function test results and see a disproportionate decease in diffusion capacity when compared to what one would expect from COPD alone and that one possible explanation for such disproportionate decrease is the presence of asbestos-related scarring. *Id.* at 65-66.

Dr. Kern

The final causation opinion is from Kenneth A. Kern, M.D., a board-certified general surgeon and oncology fellow, who reviewed the Decedent's medical records as well as the reports from Drs. Daum, Pulde and Teiger. He discussed his findings in a report dated January 9, 2004 and at a deposition taken on January 16, 2004. EX 2. His diagnosis was (1) poorly-differentiated adenocarcinoma of the left lower lobe induced by extensive cigarette smoking, (2) localized asbestos-related pleural plaque disease, (3) no evidence of asbestos-related fibrotic

parenchymal disease and no evidence of asbestos-related parenchymal fibrosis or scarring, and (4) significant bullous emphysema secondary to a 60-75 pack year smoking history. *Id.*, Dep. Exhibit 2 at 1-2. Dr. Kern observed no findings of pulmonary changes consistent with asbestosis in his review of the chest x-ray and CT scan reports, and he stated that "there is no association between pleural plaque disease, without radiographic signs of asbestosis, and an increased incidence of lung cancer." *Id.* at 4. He cited three studies between 1984 and 2002 which have shown no significant increase in lung cancer incidence among individuals with asbestos-related plaque disease but no evidence of asbestosis. *Id.* Dr. Kern wrote that a study by Hillerdahl *et al*. of 1,596 persons between 1963 and 1985 reported an increased risk of lung cancer in patients with plaque-only disease, but he pointed out that the conclusions of this study have been criticized and found non-reproducible for several reasons. *Id.* at 5, 35, 55.

At his deposition, Dr. Kern testified that his practice consists of cancer surgery, primarily breast but also melanoma, and he is in the process of completing a curriculum leading to a master's degree in public health at the University of Connecticut where he has conducted studies of cancer etiology including etiology of cancers induced by foreign body carcinogens, of which asbestos is one. EX 2 at 6-7. Specifically, he has conducted a study into whether whether silicone breast implants can cause cancer. *Id.* at 15-16. He further testified that based upon his review of the Decedent's medical records, it is his opinion that the Decedent's lung cancer was not precipitated, hastened, aggravated or accelerated by his exposure to asbestos. *Id.* at 5-6. Dr. Kern explained the differences between pleural plaques and asbestosis and said that while there is an increased incidence of lung cancer in smokers who have asbestosis, the same connection does not hold for smokers who only have pleural plaques. *Id.* at 9-10. Regarding radiographic evidence of asbestosis, Dr. Kern testified he would expect to see the following findings in the x-ray and CT scan reports:

> linear streaking, often at the lung bases and straight lines or linear lines at the periphery of the lung which is contracting and making the patterns of the peripheral airway in the lung abnormally linear. They can also see certain nodularity throughout the lung fields, which again are thickening of the bronchials and scar formation. These were not seen in this patient.

Id. at 11. He said that asbestos exposure causes mesothelioma and can contribute to the development of lung cancer under certain conditions. *Id.* at 17. He explained that in determining whether a patient's lung cancer is related to asbestos exposure, he considers whether the patient's situation fits within what has been shown in the epidemiological studies. *Id.* at 24. That is, he looks in the medical reports for radiographic evidence of asbestosis or asbestos-related fibrosis rather than pathology evidence because the epidemiological studies examined individuals with radiographic evidence of asbestosis. *Id.* at 25-26. In this regard, he stated that he does not read the x-ray films himself because he is not a certified reader and that he would defer to the interpretation of a B-reader. *Id.* at 38-39. Thus, Dr. Kern stated that it is his understanding from the literature that asbestos-related lung cancer is not a scar cancer but rather that the epidemiological studies show that there is an association between development of

radiographic evidence of asbestosis and development of lung cancer. *Id.* at 26. He continued that scar tissue is caused by chronic inflammation due to asbestos exposure and that inflammation predates the development of scarring and can be present simultaneously with scarring. *Id.* at 26-27. He further testified that inflammation results from the body's attempt to expel asbestos fibers and "[d]epending on the number of fibers and the host vulnerability and the host factors, one could develop the carcinoma." *Id.* at 27. Dr. Kern agreed that asbestos exposure causes the "K-ras oncogenic" molecular changes described by Dr. Daum which would not be visible on an x-ray, but he added that many "metaplastic" changes occur without producing malignancies and that there is a question in his mind as to when the K-ras oncogenic changes occur in relation to the development of lung cancer. *Id.* at 27-28. He said that although he would want to see a finding of asbestosis before concluding that cigarette smoking and asbestos exposure synergistically produced a lung cancer, one does not need to find emphysema or radiographic evidence of smoking-related changes before attributing lung cancer to smoking because cigarette smoke, unlike asbestos which is a "foreign body carcinogen, is a "chemical" cause of cancer or "completely carcinogenic mechanism." *Id.* at 39-40. He agreed that asbestos fibers absorb or act as a platform for heavy metals contained in cigarette smoke, but he held to his view that the asbestos deposited in the lungs can be implicated as a factor in cancer development only when it is present in sufficient quantities produce radiographic evidence of asbestosis. *Id.* at 40-41. Dr. Kern also agreed that inhalation of asbestos triggers a lung response which, in turn, results in inflammation and scarring and that scarring or fibrosis, even when seen only at the microscopic level and not radiographically, meets the definition of asbestosis. *Id.* at 42-43. However, he reiterated that the epidemiologic studies that he relies on to determine whether there is an association between asbestos and lung cancer involved patients with radiographic, not microscopic, evidence of asbestosis. *Id.* at 43.

The foregoing discussion of the expert opinions reveals sharp disagreement on whether the synergistic relationship between asbestos exposure and cigarette smoking in contributing to the development of lung cancer exists in the absence of radiographic evidence of asbestosis. On the other hand, all of the experts are in agreement that the Decedent's lung cancer would be attributable, at least in part, to his asbestos exposure if there is radiographic evidence that he had asbestosis. A review of the case law reflects that this debate is not uncommon in claims brought under the Act where asbestos exposure is alleged to have contributed to lung cancer. Neither view has prevailed as a matter of law, and the Benefits Review Board has affirmed ALJ decisions crediting both sides of the controversy as proper exercises of an ALJ's discretion to weigh and credit evidence. *Compare, e.g.*, *Sistrunk v. Ingalls Shipbuilding, Inc*., 35 BRBS 171, 173-174 (2001) (affirming denial of benefits based on ALJ's decision to credit medical opinions that lung cancer cannot be attributed to asbestos exposure in the absence of asbestosis) with *Buskey v. Ingalls Shipbuilding, Inc*., BRB Nos. 02-0564 and 02-0652 (Mar. 24, 2003) (unpublished), slip op. at 5-6 (affirming award of benefits and finding no error in ALJ's decision to credit medical opinion that diagnosis of asbestosis is not a prerequisite to finding that asbestos exposure contributed to development of lung cancer). In this case, it is not necessary to resolve the conflict in the medical opinions as to whether lung cancer can be attributed to asbestos exposure in the absence of evidence of asbestosis because I find Dr. Daum's opinion that the

Decedent did in fact have radiographic evidence of asbestosis to be better reasoned and better supported by the record than the contrary opinions from Drs. Teiger, Pulde and Kern.

First, and not insignificantly, Dr. Daum is the only expert who is certified as a B-reader. Dr. Teiger's *curriculum vitae* indicated that he had B-reader certification in the 1980s, but this was long before he interpreted the Decedent's films, and I do not consider lapsed credentials to be the equivalent of Dr. Daum's current certification especially where NIOSH requires recertification every four years. *See In Re Joint Eastern and Southern Districts Asbestos Litigation*, 237 F.Supp.2d 297, 307-308 (E.D.N.Y. 2002). Moreover, Drs. Pulde and Kern did not personally read the x-ray and CT scan films, relying instead on the reports in the record, and Dr. Kern appropriately acknowledged that he would defer to the interpretation of a B-reader which is consistent with established Board precedent that an ALJ "may assign heightened weight to the interpretations by physicians with superior radiological skills." *Zeigler Coal Co. v. Director, OWCP*, 326 F.3d 894, 899 (7th Cir. 2003). Second, Dr. Daum additionally based her asbestosis diagnosis on an interpretation of the Decedent's pulmonary function studies, as did Dr. Teiger, but I find that her explanation of how the Decedent's pulmonary function findings are consistent with the presence of both COPD and restrictive asbestosis because the two conditions tend to negate each other in terms of total lung capacity measurements to be more thorough and, consequently, more persuasive than the testimony of Drs. Teiger, Pulde and Kern on this point. Third, and perhaps, most importantly, Dr. Daum's finding of asbestosis is better supported by the objective evidence of record. As discussed above, Dr. Teiger found no "convincing evidence for asbestos-related disease" based in part on his understanding that the January 13, 2003 CT scan did not show pleural plaques or diffuse fibrotic changes suggestive of asbestosis. EX 1, Dep. Exhibit 1 at 5. He also opined that the pulmonary fibrosis observed on the Decedent's chest x-rays could be explained by the Decedent's radiation and is not typical of the diffuse fibrosis seen in cases of asbestosis. EX 1 at 52-53. While Dr. Teiger is right that the January 13, 2003 CT scan report does not mention pleural plaques, he appears to have overlooked the radiologist's findings of "biapical pleural thickening and pulmonary fibrosis." CX 3 at 11. He similarly appears to have overlooked the fact that CT scans conducted between 1998 and 2002, *before the Decedent underwent radiation therapy for his lung cancer*, showed calcified pleural plaques, "diffuse areas of fibrosis" and "linear fibrotic changes." CX 2 at 12-16, 27, 36.[7] Dr. Pulde found no evidence to support a diagnosis of asbestosis, but he somewhat grudgingly conceded the "possibility" of asbestosis if the Decedent had a sufficient exposure history, a chest x-ray classified as 1/1 under the I.L.O criteria and pulmonary function tests showing restrictive disease and reduced diffusion capacity. EX 3 at 29. Given the agreement among all of the experts that the Decedent had a significant history of exposure to asbestos, Dr. Daum's 1/1 B-reading consistent with asbestosis and her credited interpretation that the pulmonary function test results show restrictive disease co-existent with COPD, Dr. Pulde's conclusion that there is *no evidence* that the Decedent developed asbestosis is untenable. Lastly, Dr. Kern seems to have missed evidence in the record of the type that he said that he would expect to see in a case of asbestosis. That is, he testified that he would like to see "linear

[7] The Decedent underwent radiation treatment between December 13, 2002 and January 9, 2003. CX 3 at 2.

streaking, often at the lung bases and straight lines of linear lines at the periphery of the lung . . . [and] certain nodularity throughout the lung fields, which again are thickening of the bronchials and scar formation." EX 2 at 11. Had Dr. Kern carefully read the radiologist's report of the July 26, 2002 pre-radiation CT scan with contrast, he would have seen evidence of a "streaky opacity" posteolaterally in the right apex "suggestive of scarring", "[s]ome streaky opacity . . . bridging the two nodular densities" and "nodular thickening" observed in the lower left lobe, "a streaky nodular opacity posteriorly in the right lower lobe . . . more suggestive of scarring" and "some nodularity seen at the distal aspect of the major fissure on the right as it inserts in the diaphragm . . . suggestive of scarring." CX 2 at 15. He also would have seen the radiologist's finding of "linear fibrotic changes" in the report of the August 27, 2002 pre-radiation CT with intravenous contrast. *Id.* at 11. In my view, Dr. Kern's failure to reconcile his opinion with this evidence erodes the reliability of his conclusions. For these reasons, I find that the weight of the evidence establishes that the Decedent suffered from asbestosis caused, at least in part, by his exposure to asbestos during the course of his employment in EBC's shipyard.

Based on my finding that the Decedent suffered from asbestosis and the unanimous opinion of the medical experts that lung cancer is causally related to the synergistic action of both cigarette smoke and asbestos exposure when asbestosis is present, I conclude that the Claimants have carried their ultimate burden of proving by a preponderance of the evidence that the Decedent's lung cancer and death were causally related to his employment at EBC. Accordingly, the Claimants are entitled to benefits under the LHWCA.

D. Benefits Due

The Claimants seek disability compensation, funeral expenses, death benefits, medical expenses and attorney's fees.

1. Disability Compensation

Since the Decedent voluntarily retired before his asbestos-related lung cancer became manifest, his disability compensation is calculated under section 8(c)(23) of the LHWCA based on the degree of permanent physical impairment rather than economic factors. *See Frawley v. Savannah Shipyard Co.*, 22 BRBS 328, 330 (1989); 20 C.F.R. § 702.602. The parties have stipulated that the applicable NAWW for any benefits is $498.27, and they have stipulated that that the Decedent had a 100 percent disability as of September 24, 2002, the date of his diagnosis of lung cancer, which is consistent with rulings of the Benefits Review Board. *See Cormier v. General Dynamics Corp*., 30 BRBS 189, 193 (1996). Accordingly, I find that the Decedent was permanently disabled from September 24, 2002 until his death on September 20, 2003, and I will award his estate permanent partial disability compensation for this period pursuant to section 8(c)(23). The applicable compensation rate of $332.18 is calculated by multiplying two-thirds of the stipulated NAWW ($498.27) by the 100% impairment rating for the Decedent's lung cancer.

2. Death and Survivor's Benefits

As a surviving spouse who was married to and living with the Decedent at the time of his work-related death, I find that the Widow is entitled to death benefits and funeral expenses as provided by section 9 of the LHWCA. *See Griffin v. Bath Iron Works Corp.*, 25 BRBS 26, 29 (1991). The Widow introduced receipts showing that she paid a total of $5,919.54 for the Decedent's funeral. CX 8. Pursuant to section 9(a) of the LHWCA, which allows for funeral expense reimbursement up to a maximum of $3,000.00, I find that she is entitled to an award of funeral expenses in amount of $3,000.00. I further conclude that the Widow is entitled to survivor's compensation pursuant to section 9(b) of the LHWCA at the rate of 50 percent of the stipulated NAWW average weekly wage, or $249.14 per week, from September 21, 2003 and continuing until death or remarriage.

3. Interest

Interest is due on all unpaid compensation including funeral expenses. *Adams v. Newport News Shipbuilding & Dry Dock Co*., 22 BRBS 78, 84 (1989). The appropriate interest rate shall be determined pursuant to 28 U.S.C. § 1961 (2003) as of the filing date of this Decision and Order with the District Director. My order incorporates by reference this statute and provides for its specific administrative application by the District Director. The appropriate rate shall be determined as of the filing date of this Decision and Order with the District Director.

4. Medical Expenses

An employer is liable pursuant to section 7(a) of the LHWCA for those medical expenses reasonably and necessarily incurred as a result of a work-related injury. *Colburn v. General Dynamics Corp.*, 21 BRBS 219, 222 (1988); *Parnell v. Capitol Hill Masonry*, 11 BRBS 532, 539 (1979). As the responsible employer, EBC shall pay all medical expenses reasonably and necessarily incurred by the Decedent and his estate in connection with his work-related lung cancer. In addition, EBC will be ordered to reimburse the Decedent's estate for any payments already made for medical bills reasonably and necessarily incurred in connection with the Decedent's work-related lung cancer.

5. Attorney's Fees

Having successfully established their right to compensation and other benefits, the Claimants are entitled to an award of attorneys' fees under section 28 of the LHWCA. *See American Stevedores v. Salzano*, 538 F.2d 933, 937 (2nd Cir. 1976). The Claimants' attorneys have filed a an application for fees and litigation expenses, and EBC will be allowed 15 days from the date this Decision and Order is filed with the District Director to file any objection to the requested fees and expenses.

V. Order

Based upon the foregoing Findings of Fact and Conclusions of Law and upon the entire record, the following compensation order is entered:

(1) Electric Boat Corporation shall pay to the Estate of Donald E. Chappell the permanent partial disability compensation benefits to which Donald E. Chappell was entitled pursuant to 33 U.S.C. § 908(c)(23), from September 24, 2002 through September 20, 2003, at the rate of $332.18 per week, plus interest on all past due compensation at the Treasury Bill rate applicable under 28 U.S.C. § 1961 (2003), computed from the date each payment was originally due until paid;

(2) Electric Boat Corporation shall pay to the Claimant Eleanor Ruth Chappell survivor's compensation pursuant to 33 U.S.C. § 909(b) at the base rate of $249.14 per week, plus the applicable annual adjustments provided in 33 U.S.C. § 910, from September 21, 2003 to the present and continuing until death or remarriage, plus interest on all past due compensation at the Treasury Bill rate applicable under 28 U.S.C. § 1961 (2003), computed from the date each payment was originally due until paid;

(3) Electric Boat Corporation shall pay to the Claimant Eleanor Ruth Chappell funeral expenses in the amount of $3,000.00 pursuant to 33 U.S.C. § 909(a), plus interest on all such expenses at the Treasury Bill rate applicable under 28 U.S.C. § 1961 (2003), computed from the date each expense was originally due until paid;

(4) Electric Boat Corporation shall be responsible pursuant to 33 U.S.C. § 907 for reasonable and necessary medical expenses incurred by the Decedent for diagnosis and treatment of his lung cancer;

(5) The Electric Boat Corporation shall have 15 days from the date this Decision and Order is filed with the District Director in which to file any objection to the attorney's fees and expenses requested by the Claimants' attorneys; and

(6) All computations of benefits and other calculations which may be provided for in this Order are subject to verification and adjustment by the District Director.

SO ORDERED.

A

DANIEL F. SUTTON
Administrative Law Judge